Journal of Forensic and Legal Medicine 20 (2013) 345–346

Contents lists available at SciVerse ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/jflm

Case report

Toxicology observation: Nystagmus after marijuana use

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ARTICLE INFO

Article history:
Received 10 November 2011
Received in revised form
28 April 2012
Accepted 28 July 2012
Available online 1 September 2012

Keywords:
Forensic science
Marijuana abuse/detection
Humans
Psychomotor performance/drug effects
Nystagmus
Pathologic/aetiology
Substance abuse detection

ABSTRACT

Traditional teaching has held that horizontal-gaze nystagmus is a sign of intoxication by sedatives such as alcohol but not marijuana. This is a case report of an adult male who presents with 3 days of visual disturbance and dizziness following marijuana use. The exam was notable for gaze-evoked nystagmus and ataxia. Lab testing was normal except that urine drug screening was positive for marijuana only. Imaging included computed tomography (CT) and magnetic resonance imaging (MRI) scans of the head. Prior studies showing a negative association of nystagmus with marijuana are reviewed. This case is presented as a possible exception to the generalisation that marijuana is not associated with nystagmus.

1. Introduction

The Standardized Field Sobriety Tests (SFSTs)[1] are widely used by non-clinical personnel to detect clinical intoxication. They are typically used as a pre-arrest screen by law enforcement personnel, and have been validated specifically for impairment by ethanol.[2] As part of the exam, the tester directs the subject into lateral and upward gaze, observing for smooth pursuit tracking and nystagmus. The presence of nystagmus to lateral or vertical gaze or onset of less than 45° deviation is considered suggestive of intoxication. Traditional training for the SFST indicates that the presence of nystagmus at various points is suggestive of intoxication by sedatives including alcohol, barbiturates and phencyclidine (PCP), but not marijuana. The following is a description of a case in which marijuana use is associated with nystagmus.

2. Materials and methods: case report

The patient is a 25-year-old male who presented to a local emergency department with a 3-day history of persistent visual disturbance and dizziness. He noted that his symptoms were aggravated by gazing laterally or with change in head position. He also noted difficulty in coordination during daily activities such as dressing and walking. He had no prior medical or surgical problems; he was not currently using medications. His review of systems was positive for nausea, vomiting, and a limited recent headache. He denied fever, chills or tinnitus. He smoked 4 g of marijuana and drank several beers 4 days and again 3 days prior to presentation. He denied ethanol or marijuana consumption for the 2 days before presentation. He denied focal weakness or numbness, dysarthria, aphasia or other symptoms.

On exam the patient was awake, alert and appropriate. His vital signs were normal. Pertinent positive findings by neurologist's exam were left end-gaze sustained nystagmus, rotational nystagmus to vertical gaze and ataxia. The remainder of the neurologic exam was normal. Ear, nose and throat (ENT), cardiac, pulmonary and abdominal exams were normal.

All of his laboratory work was normal except for the urine drug screen, which was positive for cannabinoids and negative for PCP, amphetamines, barbiturates, benzodiazepines, cocaine and opiates. A biologic fluid screen for ethanol was not obtained. A computed tomography (CT) scan of the head and magnetic resonance imaging (MRI) scan of the head, cerebellum and brainstem were both negative for pathology. The electrocardiogram showed normal sinus rhythm with normal intervals. His symptoms markedly improved after administration of intramuscular (IM) lorazepam, to the point of becoming symptom-free and non-ataxic. Both neurology and toxicology consultants concluded that the vertigo and nystagmus were due to marijuana use. The patient was advised

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1752-928X/$ – see front matter
http://dx.doi.org/10.1016/j.jflm.2012.07.014

to discontinue its use, and was released with directions to follow with his personal physician.

3. Discussion

The oculomotor effects of marijuana were first investigated during the period when such effects by alcohol were described. Spector[3] described the absence of nystagmus as measured by electronystagmography recordings in 72 volunteers who smoked marijuana. Flom[4] and Baloh[5] described the oculomotor effects of alcohol including inhibition of smooth tracking and saccadic tracking, but neither found significant effect when marijuana was studied alone and the latter found marginal additive effect when combined with alcohol. These studies supported the conclusion that marijuana use does not cause nystagmus on the SFST. A later study[6] found impairment of smooth pursuit eye tracking with marijuana, especially at higher doses and tracking in lateral fields, but neither nystagmus nor saccadic tracking were studied. One recent study[7] found that test subjects also displayed a lack of smooth pursuit but not nystagmus after smoking marijuana. Dizziness is a well-described adverse effect of marijuana, especially in studies of the medical use of cannabinoids.[8] However, it is unusual for vertiginous symptoms to persist for several days. There have been a number of case reports of stroke or transient ischaemic attacks (TIA's) associated with heavy marijuana use, recently summarised by Thanvi and Treadwell.[9] Of the nine case series reviewed with imaging studies, eight reported positive studies for infarcts and the ninth involving TIA's reported only white-matter changes. Of interest is that the majority of the infarcts reported in this review involved the cerebellum.

Our patient had onset of dizziness and visual changes after consuming marijuana and alcohol, which persisted 2 days later. In retrospect, it would have been useful to know if other adulterants were present but the source marijuana and specific testing were not available. The association of this patient's nystagmus with marijuana would have been stronger had testing been performed to exclude the presence of ethanol. Although there are anecdotal reports of vertigo after marijuana use on the Internet, there are only two published studies relating the oculomotor effects of cannabinoid use and in these nystagmus has not been described. This case is presented as a possible exception to the generalisation that marijuana is not associated with nystagmus.

Ethical approval

None.

Funding

No financial support is reported.

Conflict of interest

None.

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